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THE LOST ART OF HEALING

PRACTICING COMPASSION IN MEDICINE

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BERNARD LOWN, M.D.
WINNER OF THE NOBEL PEACE PRIZE

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11

Digitalis: The Price of Invention

FOR MORE THAN forty years, I have engaged in cardiovascular research in addition to my clinical practice. Research enriched my knowledge of medicine and honed my scientific perspective; despite a high dosage of drudgery, experimental work has been a heady adventure. The triumphs of discovery made me feel the way I imagine a mountaineer must feel on reaching the top of a hitherto unscaled peak.

My earliest investigations focused on the then most popular heart medicine, digitalis, a drug whose introduction in 1775, by the British physician and botanist William Withering, marked the beginning of the modern era of cardiac therapy. Dr. Withering noted that an old woman herbalist in Shropshire, England, had been successful in curing dropsy, that is, edema, "after the more regular practitioners had failed." She employed twenty or more herbs, but Dr. Withering, an astute botanist, rapidly determined that the active ingredient was digitalis, found in the leaves of the purplish foxglove plant. As he ran a large clinic for poor people, he experimented freely and promptly defined the remarkable therapeutic effect of digitalis. Believing he had discovered a new diuretic, he described it in the minutest detail in his classic book, published a decade later.*

One might imagine that a drug in continuous use for more than 150 years would hold few surprises for doctors, but as a

* William Withering, *An Account of the Foxglove and Some of Its Medical Uses* (London: M. Swiney, 1985).

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young researcher in 1950, I soon realized that even experienced medical practitioners were not too clear about when and how to use this medicine. Erroneous ideas were passed, like religious writ, from generation to generation, from one medical textbook to another.

Digitalis had become the cardiologist's mainstay for good reason. First and foremost it strengthens the force of cardiac muscle contraction, thereby addressing the central problem of the failing heart, impaired pumping. Excessive fluid, sequestered in body cavities and manifest in swollen ankles, is excreted. Unhealthy weight is shed rapidly and in large amounts. Breathing is improved. The racing pulse is reined in. The patient can walk without puffing. Stretching out in bed is no longer accompanied by choking cough. For the first time in weeks the patient gets a night's sleep. The draining fatigue, which makes the smallest effort an insuperable chore, dissipates.

Not surprisingly, digitalis has been referred to as the wonder drug. The wonders, however, do not come without a price. At full therapeutic action, the patient receives a near toxic dose. Side effects, frequently insidious, are marked by a loss of appetite, dizziness, vertigo, and a discomforting fullness in the head. But the appearance of cardiac arrhythmias, some auguring death, is far more dangerous.

A distressing yet empowering experience relating to the use of digitalis occurred at the beginning of my medical career in 1950. The senior resident on the female ward at the Peter Bent Brigham Hospital asked me to see one of Dr. Samuel Levine's newly admitted elderly patients. Mrs. M. was in heart failure with congested lungs and massively edematous swollen legs. Breathing oxygen did not improve her rapid and labored respirations. One factor for the congestion was her extraordinarily rapid heart rate, a staccato 190 beats per minute. Even a healthy heart decompensates when driven three times its normal rate, but Mrs. M.'s heart had been seriously impaired by childhood rheumatic fever.

When I met Levine that morning, I urged him to see Mrs. M.

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promptly, as she was by far the sickest patient on his service. While admiring the thoroughness of Levine's history-taking and the meticulous craftsmanship of his physical examination, I was dismayed to hear him prescribe a large dose of digitoxin (one of the family of digitalis drugs) and a mercurial diuretic. Without thinking, I burst out, saying, "This combination will kill her. She will surely die within the day." Levine's eyes narrowed with ill-concealed anger. Lips barely moving, he said, "Record your opinion in Mrs. M.'s chart, exactly as you just stated." He quickly stalked away.

On finishing the brief note, I sat, my energy drained as though I had just climbed the Bunker Hill Monument. The day that had just begun dragged as if the clock had been inadequately wound while I was hoping against hope that my foolish prognostication would prove wrong. A visit to the ward late that afternoon, however, dissipated any prospect that Levine's prescription would ameliorate Mrs. M.'s perilous condition. The extra dose of digitalis had not slowed her heartbeat; on the contrary, her heart was racing even faster. Because of delays on the busy ward, she had not yet received the diuretic that I was certain would deliver the coup de grace. Panic prevented me from thinking through the options. Forty-five years ago it was unthinkable for a mere pup of a trainee to question, let alone countermand, medical orders of a senior attending physician, especially one as distinguished as Dr. Levine.

The next morning I raced to see Mrs. M., but her bed was empty. She had died during the night. The resident told me that the diuretic finished her off. As she began to pass large quantities of urine, her situation worsened by the moment. Her heart rate reached 220 beats per minute, she turned blue, gasped, and died. Resuscitation was not attempted, as the technique was not to be discovered until a decade later.

While horrified at the outcome, I confess with shame that I was more troubled about the possibility of losing my fellowship than at the tragic fate that had befallen Mrs. M. My career as Levine's fair-haired boy, so promising the day before, was seemingly at an end.

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I waited with a heavy heart for Levine's arrival, for I dreaded telling him about Mrs. M. As soon as we met, Levine asked about her. With bowed head, I whispered that she had died during the night. Levine walked briskly away, barking out, "Follow me!" I followed, feet dragging, feeling like a man about to be sentenced for an unspeakable crime. When we came to his small office, he locked the door. Looking ashen, he startled me with the words "What did I do wrong?"

Recalling the moment nearly a half century later, I am still shaken by the sizzle of intense emotions and the unexpected reversal of roles. The convict was expected to pass sentence on the revered judge. An inexperienced medical upstart was being asked to render an opinion about the improper use of digitalis by one of the great cardiologists, whose forte was clinical pharmacology. For me, this was Levine's supreme moment as a human being. Never had I encountered someone with the moral rectitude to confess such a grievous misdeed, in the process abasing himself before a young student.

I explained to Levine that Mrs. M.'s heart rhythm disorder at the time of admission was a hitherto undescribed cardiac mechanism that I had recently discovered and designated as paroxysmal atrial tachycardia with block (PAT with block). Usually, it was an expression of digitalis poisoning. In the presence of PAT with block, administering more digitalis was like attempting to extinguish a blaze by pouring gasoline on it. With each increment of drug, the heart rate accelerated, and when it reached more than 200 beats per minute, the heart abruptly catapulted into ventricular fibrillation, the mechanism of cardiac death. Having seen the arrhythmia emerge under similar circumstances and hearing Dr. Levine prescribe more digitalis, I could not contain my dread at the tragic outcome, which was made inevitable by the addition of a diuretic drug. PAT with block most commonly developed in patients who had received excessive digitalis and were then leached of body water and salts, or electrolytes, with a diuretic.

Levine listened attentively without a single interruption. When I finished, he asked a few questions and then com-

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mented, "Bernie, I appreciate your teaching me. I should have been less proud and paid attention to what you said." He never again referred to this event, but thereafter he turned to me for advice on the use of digitalis drugs in particular patients.

What Levine did not know was that I had experienced a similar tragedy as a result of my own ignorance. Sadly, my newfound expertise on the use of digitalis had also come at high price. The year was 1948, when I was a junior assistant resident (JAR) at the Montefiore Hospital in the Bronx, dedicated to chronic care. Many terminally ill patients or those with intractable chronic problems were transferred to Montefiore for long-term care from all over New York City. As a JAR, I had enjoyed almost complete autonomy. The responsibilities were enormous, and at times awesomely misplaced. Supervision was largely by senior residents, doctors in training, who had only one more year of clinical experience than I. The attending physicians, who were officially in charge, limited their oversight to the brief morning rounds. The training of doctors was accomplished at a cost to life and limb of the economically disadvantaged, for the house staff was in complete command in the wards where these patients were admitted.

There is a tendency to gild memories of the past, "the good old days." Yet reflecting on hospital care fifty years ago, I can plainly see that enormously positive changes have been wrought since. Hospitals now are far safer, patients are better informed, drugs are far more carefully dispensed, operating rooms are vastly improved. The most important development is that the patient has much more of a say in what is being done. In retrospect, the mayhem committed within hospitals five decades ago staggers the mind.

It was past midnight when a thirty-year-old woman was rolled onto my medical ward at Montefiore. Ms. W. weighed only eighty-nine pounds. She was feverish, pathetically pale, and her muscles were wasted. Her eyeballs dangled like dried prunes in largely empty sockets, her skin hung in loose dehydrated folds. Her problems, though numerous, were largely the

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result of the odd combination of anorexia nervosa and ulcerative colitis. Intractable diarrhea was draining her vital body fluids while her anorexia precluded oral replacement.

Being inexperienced, I fixated on her rapid heart rate, 170 beats per minute. Digitalis was the premier drug to contain the speeding heart. Because of the state of her gastrointestinal tract, the drug had to be given intravenously and that very week I had read about an old but extremely effective French digitalis agent, ouabain. It could be administered only by vein and was ultra-rapid acting, seemingly ideal for Ms. W. Because it was past midnight, no one was around to offer me guidance.

Judging her situation critical, I hooked up an electrocardiogram and injected in her one remaining vein a bolus of what I thought was a small dose of ouabain, one-fifth the usual dose. Nothing happened for five minutes. Then abruptly Ms. W. began to flail her arms and thrash around like a fish out of water. Her mouth grimaced monstrously, repeatedly opening in a wide yawn as though she was hungering for air. Instead of slowing, the heart kept speeding. Within several minutes she turned a ghastly shade of purplish blue. As I looked down on the strips of electrocardiographic paper, the rhythm turned chaotic, proclaiming the death knell pattern of ventricular fibrillation. Small bundles of wasted muscle twitched agonally, grasping for the last molecule of oxygen from the no longer circulating bloodstream. Transfixed into helplessness, I stood quavering as though watching a murder committed on a 3-D movie screen. Ms. W. was dead within eight minutes of receiving the injection.

The next morning at medical report, guilt, coupled with a lack of sleep, enhanced my throbbing sense of foreboding. Without omitting any details, I bluntly told the medical staff what had happened and showed them the remarkable electrocardiographic strips. I craved punishment, yet no one faulted my judgment or behavior. On the contrary, I was treated with the respect accorded a heroic soldier in the trenches. The only comments were: "Don't take it so hard," "Win some, lose some," "This is the price you have to pay to become a doctor,"

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"To gain experience you have to take a few hard knocks," and other such meaningless platitudes meant to show solidarity.

The most galling comment was "You took a calculated risk." Yes, I did the calculating, but the patient took the risk without having been given a chance or choice. The chief of medicine, a kindly fatherly figure, consoled me with the words that good judgments come from experience, "but experience is the name we give to bad judgments."

It was unreal! A summary execution had taken place but no moral tremor was felt. Granted, the action was not premeditated and it arose out of ignorance, but when is ignorance a mitigating justification for such a transgression? Within five minutes after I had related the tragic event, everyone turned to the next case without the slightest trickle of indignation. A patient had been prematurely dispatched to the hereafter, and these very well-trained doctors, who were good human beings, had commiserated with the physically scratched culprit, apparently indifferent to his dead victim.

A week later, when the patient's blood chemistries returned, I was startled at the profound derangement in electrolytes, including potassium, sodium, and chloride. I did not immediately connect the extremely low potassium, only 1.6 milliequivalents, about one third of the normal concentration, with her extreme sensitivity to the digitalis drug ouabain. The relation between potassium and digitalis had not yet been discovered.

Later that year, when I knew more, I reexamined the patient's electrocardiogram. It was clear that on admission her heart rhythm had been sinus tachycardia, a fast heartbeat, the normal physiologic response to stress either physical or psychological. In such a clinical situation, digitalis is ineffective in slowing the tachycardia. This still did not account for her striking sensitivity to digitalis. The patient had not received any digitalis before the fatal small dose and the postmortem examination was unrevealing, showing a perfectly normal heart muscle, intact heart valves, and widely patent coronary vessels. In the absence of heart disease, even a massive overdose of digitalis, while it can

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poison, does not kill. According to the knowledgeable chief cardiologist at the Montefiore Hospital at the time, young people with normal hearts could consume buckets of the stuff. In searching the published literature, I came across someone who, in a suicide attempt, took 200 times the dose I had administered and survived. Why then this marked sensitivity?

I had little time to sleuth for an answer. House staff training was arduous, with duty on alternate nights and alternate weekends. The patient load was high, with many of them on the verge of death. Arriving home weary, I would find that my two young children were hungering for fatherly attention, and my wife, cooped up in a small apartment, away from friends and family, was starved for companionship. I had little chance to brood or obsess about my fatal error, and the unsettling experience sank to the depths of my consciousness but continued to exert a magnetic subliminal force.

The following year, as senior resident, I had more time to reflect, and the sad Ms. W. began to reappear in my thoughts when I was trying to relax or before I fell asleep. Like a toothache constantly tongued into consciousness, the woman's death haunted my mind, the memory of her stirred up by the numerous patients I saw with digitalis intoxication. Many of them were receiving a fixed dose of digitalis, and a toxic state was provoked by the administration of a mercurial diuretic. No sooner had the copious flow of urine stopped than the patient began to feel wretched with nausea, vomiting, dizziness, and weakness. The heartbeat grew irregular from salvos of ventricular extrasystoles. In sick elderly patients, digitalis toxicity was a particularly ominous condition, commonly auguring death.

In the mid-1930s, Levine had provided the most widely accepted explanation for diuretic-induced digitalis intoxication. He reasoned that when a diuretic acts on the kidney to rid the body of excess salt and water, the fluid cannot reach the kidney without traversing the heart. Levine surmised that since digitalis was distributed throughout the body, fluid mobilized by the diuretic contained substantial quantities of the drug. Diure-

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sis was therefore equivalent to administering more digitalis by redistribution from body stores. Levine was convinced that he had proved his hypothesis when he demonstrated that edema fluid leached from patients taking digitalis contained enough of the drug to stop a beating frog heart. In short, the cause of the digitalis poisoning induced by administering a diuretic was due to heart muscle being exposed to more digitalis, which Levine designated as diuretic redigitalization.

This theory had little relevance to Ms. W.'s case. She had never been given diuretics, and before receiving ouabain had not been exposed to digitalis drugs. Some other mechanism must be found to account for her supersensitivity to digitalis. This conclusion stimulated me to examine Levine's hypothesis more critically. According to his theory, digitalis poisoning should occur only in patients with sizable diuresis, while those with meager urinary outputs would not experience this redigitalization phenomenon. The ugly fact was that I could detect no such clear-cut relationship. At times patients who barely voided in response to the diuretic appeared massively poisoned; others who lost gallons of fluid, weight loss of ten or more pounds, showed not a scintilla of digitalis overdose.

To test Levine's theory, I administered extra doses of digitalis to patients who had experienced digitalis poisoning after a diuretic. The dose I prescribed far exceeded the amount that Levine suggested could be present in the fluid secreted after the diuretic. Nonetheless, not a single patient developed digitalis poisoning from that single additional dose. They did not experience any of the typical symptoms nor did they develop the telltale arrhythmias of overdigitalization. For me, Levine's hypothesis was demolished. But I had not identified the X factor that was being washed out of the body, which accounted for the increased sensitivity to digitalis.

If the diuretic was washing something out of the body, what could it be? Ms. W.'s case, like Banquo's ghost in *Macbeth*, hinted at the answer. The haunting refrain to her abrupt death crystallized into a question: Does low body potassium affect cardiac sensitivity to digitalis? Searching the medical literature,

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I found that a San Francisco cardiologist, John Sampson, discovered in the early 1930s that potassium could abolish ventricular extrasystoles, or extra heartbeats, a frequent expression of digitalis overdosage. But this left unanswered the essential question about the potential adverse relationship between low potassium and digitalis drugs.

If potassium was the X factor, then a reduced concentration of blood potassium should be found only in those who had symptoms of digitalis poisoning after receiving a diuretic and not in those without such symptoms. To my delight this proved to be the case. But my concept had not yet been proved with the rigor demanded by science. To do so it was necessary to replicate experimentally what happened to Ms. W., specifically demonstrating that a diuretic drug sensitized the heart to digitalis only when it leached out substantial amounts of body potassium, thereby resulting in an abnormal blood concentration of potassium, a condition designated as hypokalemia. If my hypothesis was correct, sensitivity to digitalis after a diuretic should be a function of the extent to which the blood potassium was lowered by the diuretic.

The experiment was easier to describe than carry out. How could I show that a patient who had not been receiving digitalis became more sensitive to the poisonous action of digitalis by virtue of a diuretic-induced loss of body potassium? This seemed like an insoluble conundrum. Then, as frequently transpires, the insoluble became straightforward. Again the memory of Ms. W. suggested an answer. Why not use a short-acting digitalis drug like the one I gave her to titrate the dose required for provoking mild toxicity? If my view was correct, patients who had lost potassium after a diuretic would require substantially less digitalis to reach a toxic endpoint. The experimental design was now apparent: administer a short-acting digitalis drug to patients before and after a diuretic. For this I needed a safer drug than ouabain, more rapid in onset of action and more prompt in total dissipation of cardiac action.

I searched all the different forms of rapid-acting digitalis drugs available, but none fit the bill. Then I learned that Dr.

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Charles Enselberg, a cardiologist at the Montefiore, was experimenting with a newly synthesized digitalis agent, acetyl strophanthidin. This drug, a first cousin to ouabain, could be given to a full digitalizing dose within a couple of minutes and was totally eliminated from the body within less than two hours. Overdosing could not lead to serious toxicity, since the drug was rapidly dissipated. It was ideal for the study I had in mind. At the time there were no human-use committees to rule on what was permissible to do to patients. No informed consent was required. All that was necessary was to ask permission from the chief of service. The answer was invariably in the affirmative. In fact, one gained brownie points by showing a penchant for research.

My enthusiasm waxed day by day, my emotions churning at high speed. I rapidly recruited ten patients with heart failure who had not yet been given digitalis and gave them by vein the amount of acetyl strophanthidin they would tolerate. Two hours later, when the titration was completed and all the drug was eliminated from the body, they received a mercurial diuretic. Over the next twenty-four hours, every drop of their urine was collected to measure their excretion of potassium. The following day I again titrated their tolerance for acetyl strophanthidin. Some patients took much less drug to reach toxicity the second time, while others tolerated exactly the same dose. In order not to bias results, I asked Dr. Ray Weston, a research cardiologist, to analyze the data. When he showed me the final outcome, I could not contain my elation. The patients who showed no change had lost no potassium and their blood potassium remained unaltered. On the other hand, those who during the second administration of acetyl strophanthidin were more sensitive and could tolerate far less drug had lost large amounts of potassium in the urine. This was corroborated by a marked decrease in the blood potassium concentration.

Without any fiscal support or technical assistance, I was forced to do all the scut work. The investigation did not free me from the heavy clinical load as resident. It meant that my fourteen-hour working day was extended by several hours, but

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the excitement of the work, the important clinical implications, revved up extraordinary energies. The drudgery and sleepless nights were well worth it. The research resulted in my first medical publication. Meager as the article was, a mere four pages, it was widely noted and cited. The work was editorialized in the *Journal of the American Medical Association* as well as in the leading British medical journal, *Lancet*. Both deemed it a milestone study.

Digitalis poisoning was a leading cause of fatality among patients with heart failure, and my discovery that diuretic-induced loss of potassium sensitized the heart to the toxic action of the digitalis drugs raised numerous questions that urgently needed to be answered. To continue the investigations it was important to obtain a cardiovascular fellowship in a leading research hospital. The Peter Bent Brigham Hospital (PBBH), now the Brigham and Women's Hospital, was the ideal institution in which to investigate the full ramifications of the digitalis-potassium relationship.

At the time, Dr. John Merrill was carrying out pioneering studies at the PBBH with the recently introduced artificial kidney, and hundreds of patients were being dialyzed annually. Many were in heart failure and receiving digitalis. Dialysis, in addition to removing metabolic waste products, was washing out potassium from the body. This offered a unique opportunity to determine expeditiously how removing body potassium affected patients receiving digitalis drugs.

A more profound attraction was the opportunity to work with Dr. Levine, though there was little if any chance of my gaining a fellowship with him. He took only one person for a single year and favored Harvard graduates, especially those who had matriculated through the PBBH house staff. Nothing was to be lost by applying, however, and to my surprise I was granted an interview.

I devised a bizarre strategy. From the medical grapevine I learned that Levine was impeccably honest and exceedingly proud and that he did not take kindly to being contradicted. I therefore decided to challenge his work. When I presented my

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unimpressive credentials, he asked, as anticipated, why I wished a fellowship with him. I answered that my aim was to prove him wrong on an important concept he had been promulgating. Momentarily startled, he inquired what that might be. I explained that his widely accepted theory about diuretic redigitalization was erroneous. He was barely able to restrain himself. "You obviously have not read our paper on this subject, because we proved with certainty in a frog heart preparation that edema fluid has large quantities of digitalis."*

I replied with a carefully prepared argument about the critical role that I had discovered for potassium, adding that the PBBH was the ideal place for me to continue my research. Then came my well-rehearsed punch line, namely, that every good scientist, like Levine, believes that next to the promulgation of truth, the public recantation of error was the greatest virtue. Levine's hostile demeanor when we parted indicated that there was not the remotest chance of my procuring the appointment. But Levine proved himself to be a man of principled integrity. Although his ego was bruised, he nonetheless offered me a job, though he duly punished me for my chutzpah by not permitting me to work on digitalis for more than a year.

But in my second year, Levine freed me to work on the drug. Within short order, we decisively proved the digitalis-potassium relationship. It alerted doctors to the importance of potassium loss in the genesis of digitalis intoxication and heart arrhythmias. It led them to prescribe supplementary potassium when administering diuretics and stimulated introduction of a new class of potassium-sparing diuretics. Our findings were summarized in a number of publications and in a book that became a medical best seller. Barely thirty years old, I was catapulted into a world authority on the most important heart drug at the time. During the 1950s, the subject of my extensive lecturing was exclusively "The Use and Abuse of Digitalis." Once, while introducing me as the guest speaker at a medical

* M. A. Schnitker and S. A. Levine, "Presence of Digitalis in Body Fluids of Digitalized Patients," *Archives of Internal Medicine* 60 (August 1937): 240–250.

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meeting in a small city in Michigan, the chairman became flustered and spoke approximately as follows: "It is a great privilege for our hospital to be addressed by Dr. Lown, the world's leading digitalis poisoner." Suddenly, Ms. W. in her terminal agony appeared before my eyes. Guilt gnawed like an ulcer.

The work on digitalis and potassium led to my lifelong appreciation of every drug as a potential poison in disguise, which was hardly a novel observation. Paracelsus, the medieval alchemist and physician, wrote, "All substances [drugs] are poisonous; there is none that is not a poison. The right dose distinguishes a poison and a remedy."

As he usually did, Shakespeare stated it more poetically. In *Romeo and Juliet,* Friar Lawrence searches for a potion to put Juliet in a sleep simulating death. On encountering a flower with medical powers, he reflects:

> Virtue itself turns vice, being misapplied;
> And vice sometime's by action dignified.
> Within the infant rind of this small flower
> Poison hath residence, and medicine power.

My research work on digitalis led to a number of further significant advances. It aroused interest in cardiac arrhythmias, since digitalis drugs can provoke every conceivable heartbeat derangement. For the first time, doctors learned that atrial rhythm disorders could follow excess digitalis. The research also led to the abandonment of long-acting digitalis drugs like digitalis leaf, digitoxin, Gitaligin, and other such preparations. My agitation for the use of digoxin led to its universal acceptance, and legions of lives have been saved through the safer use of the digitalis drugs.

Although in death Ms. W. left a legacy of life, I remain convinced that good ends rarely justify bad means. The tragedy I inflicted on Ms. W. occurred early in my life and honed my moral sensibilities in confronting the myriad of problems a doctor faces. I became sensitized to the fact that far too often in medicine, as in the world at large, reasonable goals propel evil

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deeds. Foul means often pollute good ends. Few would contest that prolonging life is an unquestioned good. Yet in aiming to prolong life, health professionals frequently inflict indescribable misery. Worse still is the rationalization that good intentions provide sufficient sanction for questionable acts. I am persuaded that the moral fiber frays when means and end are sundered.

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